confirmed the diagnosis of donovanosis. A 6 week course of doxycycline 100 mg twice daily was commenced, and the patient made a full recovery.

In view of the increased transmissibility of HIV in association with ulcerating genital infections, an effective and acceptable oral agent is required for the treatment of donovanosis. Few well designed controlled trials have addressed this problem.[2] Effective treatment and prevention of ulcerating genital disease is important in the control of the spread of HIV. Clinicians investigating patients from tropical countries should be aware that trimethoprim and co-trimoxazole do not always eradicate donovanosis.

KARL BIRTHISTLE
JAMES GREIG
Department of Medical Microbiology, St George's Hospital Medical School, London SW17 0RE
PHILIP HAY
Department of Genito-urinary Medicine, St George's Hospital Medical School, London SW17 0RE

1 Merianos A, Gilles M, Chuah J. Ceftriaxone in the treatment of chronic donovanosis in central Australia. *Genitourin Med* 1994;**70**:84–9.
2 Richens J. The diagnosis and treatment of donovanosis (granuloma inguinale). *Genitourin Med* 1991;**67**:441–52.
3 Garg BR, Lal S, Sivami S. Efficacy of co-trimoxazole in donovanosis. *Br J Venereal Dis* 1978;**54**:348–9.
4 Lal S, Garg BR. Further evidence of the efficacy of co-trimoxazole in granuloma inguinale. *Br J Venereal Dis* 1980;**56**:412–3.
5 O'Farrell N. Clinico-epidemiological study of donovanosis in Durban, South Africa. *Genitourin Med* 1993;**69**:108–11.
6 O'Farrell N. Global eradication of donovanosis: an opportunity for limiting the spread of HIV-1 infection. *Genitourin Med* 1995;**71**:27–31.
7 Pradinaud R, Grossbans E, Basset A, Bertin C. Etudes de 24 cas de donovanose en Guyane Française. *Bull Soc Pathol Exot Filiales* 1981; **74**:30–6.
8 Anonymous. Co-trimoxazole use restricted. *Drug Ther Bull* 1995;**33**:92–3.

Accepted for publication 12 February 1997

Perforation of hard palate in lues maligna associated with HIV infection

Unusual oral and skin manifestations of infectious diseases may be observed in patients with HIV infection.[1-3] These are quite often a challenge to the clinician. Destructive bone diseases such as osteitis and osteomyelitis are well known complications of congenital and tertiary syphilis; they are rare complications of early acquired syphilis.[4] We report a case of perforation of hard palate in lues maligna associated with HIV infection.

A 30 year old promiscuous male presented with a solitary genital ulcer and recurrent erythematous nodules and ulcers over the limbs (fig 1) and trunk for the past 5 months, associated with joint pains, swelling over the limbs, fever, headache, myalgia, and epistaxis. On further examination he had inguinal and epitrochlear lymphadenopathy. Oral examination revealed a mucosal patch and perforation over the hard palate (fig 2). Dark ground microscopy from the genital ulcer revealed several motile spirochaetes. VDRL was reactive in 1:128, TPHA and HIV (ELISA) were positive. Blood examination revealed normocytic normochromic anaemia with increased rouleaux formation, raised ESR (> 150 mm in the first hour). Liver function tests, antinuclear antibody (ANA), and double stranded DNA were within normal limits. The patient refused to undergo skin biopsy and lumbar puncture. In view of the clinical features, dark ground examination, and serological findings secondary syphilis (lues maligna) was considered. He was treated with procaine penicillin 24 units for 2 weeks. On follow up his skin lesions had healed.

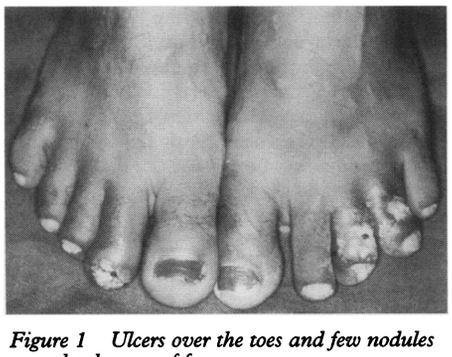

Figure 1 Ulcers over the toes and few nodules over the dorsum of feet.

The case is interesting because of early palatal perforation following lues maligna in this HIV patient. A review of the literature of the past 20 years revealed that of 1800 patients with early syphilis, less than 0·2% had evidence of periostitis, and there were no reports of destructive bone lesions.[5] A recent review of bone and joint disease in association with HIV infection does not report syphilis related bone disease.[6] Cases of HIV associated lues maligna with widespread, atypical ulcerations of the oral mucosa and skin have been reported.[7-9] We postulate that defective cell mediated immunity might have facilitated the rapid dissemination of spirochaetes invading bones and joints resulting in bone destruction. Numerous spirochaetes observed in bone biopsy specimen suggesting the aggressiveness of syphilis in a patient with concurrent HIV infection has been documented earlier.[10]

C BALACHANDRAN
L SABITA
G R KANTHRAJ
Department of Skin & STD, Kasturba Medical College, Manipal—576 119, Karnataka, India

Correspondence to: Dr C Balachandran.

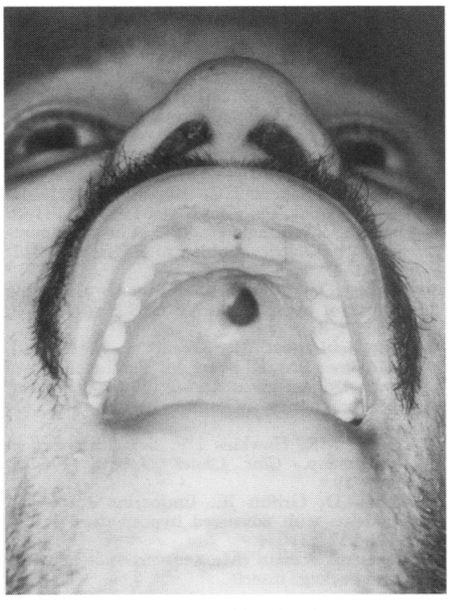

Figure 2 Perforation of hard palate.

1 Ficarra G, Shillitoe EJ. HIV-related infections of the oral cavity. *Crit Rev Oral Biol Med* 1992;**3**:207–31.
2 Ficarra G. Oral lesions of iatrogenic and undefined etiology and neurologic disorders associated with HIV infection. *Oral Surg Oral Med Oral Pathol* 1992;**73**:201–11.
3 Penneys NS, Hicks B. Unusual cutaneous lesions associated with acquired immunodeficiency syndrome. *J Am Acad Dermatol* 1985;**13**:845–52.
4 Dismukes WE, Delgado DG, Mallernee SV, Myers TC. Destructive bone disease in early syphilis. *JAMA* 1976;**236**:2646–8.
5 Mindel A, Tovey SJ, Timmins DJ, Williams P. Primary and secondary syphilis, 20 years experience. *Genitourin Med* 1989;**65**:1–3.
6 Hughes RA, Rowe IF, Shanson D, Keat AC. Septic bone, joint and muscle lesions associated with human immuno-deficiency virus infection. *Br J Rheumatol* 1992;**31**:381–8.
7 Shulkin D, Tripoli L, Abell E. Lues maligna in a patient with human immunodeficiency virus infection. *Am J Med* 1988;**85**:425–7.
8 Glover RA, Piaquadio DJ, Kern S, Cockerell CJ. An unusual presentation of secondary syphilis in a patient with human immunodeficiency virus infection: a case report and review of the literature. *Arch Dermatol* 1992;**128**:530–4.
9 Ficarra G, Zaragoza AM, Stendordi L, Parri F, Cockerell CJ. Early oral presentation of lues maligna in a patient with HIV infection. *Oral Surg Oral Med Oral Pathol* 1993;75:728–32.
10 Kastner RJ, Malone JL, Decker CF. Syphilitic osteitis in a patient with secondary syphilis and concurrent human immunodeficiency virus infection. *Clin Infect Dis* 1994;**18**: 250–2.

Accepted for publication 12 February 1997

Sharing prescribing of continuous aciclovir treatment: effects of a new policy and general practitioner responses

Recent guidelines on the care of patients with genital herpes[1] include reference to improved communication between local genitourinary medicine clinics and general practitioners. This reinforces anecdotal reports that in some areas of the UK general practitioners and genitourinary medicine clinics were satisfactorily collaborating in the management of patients requesting continuous treatment with aciclovir.

In early 1996 the national reduction in HIV funding by 7·7% and the lack of designated additional funding for new antiretroviral drugs have resulted in extreme pressure on the drug budget within the directorate of HIV/GU medicine at the Chelsea and Westminster Hospital and prompted us to examine ways of extending collaboration with general practitioners with the secondary gain of achieving an overall reduction in our drugs budget. After discussion within the unit and with purchasers we attempted to reduce our projected deficit by restricting hospital prescribed therapies to treatments for HIV infection, and acute and new presentations of STDs. With the intention of preserving patient choice concerning confidentiality, however, we asked for our patients' specific consent to write to their general practitioners to request them to share responsibilities in the prescribing of continuous aciclovir for the prevention of recurrent genital herpes.

This policy took effect from April 1996, and during the following 5 months we approached 71 patients who were receiving regular prescriptions for continuous aciclovir to prevent recurrences of culture proved genital herpes from the genitourinary medicine clinic. Eight patients refused to have their herpes diagnosis and prescription needs disclosed to their general practitioners,

all as a result of concerns about confidentiality. All these patients have documented their decision in writing. One patient offered to make a contribution to the costs if it was possible to continue on hospital prescriptions, but there is at present no logistical method for achieving this. Of the 63 general practitioners contacted, four refused to prescribe the recommended aciclovir treatment, predominantly citing the grounds of costs or not wishing to accept responsibility for the care. Overall, 59 patients were successfully referred to their general practitioners who continued to prescribe the recommended dosages of aciclovir. This did not reduce the frequency of visits to the genitourinary medicine clinic as continued monitoring of the therapy, ongoing support, and counselling and discussion on when to cease therapy were carried out in the clinic according to our protocol.

Our experience suggests that the overwhelming majority of patients are prepared to give permission for the involvement of the general practitioner in the management of their genital herpes and that the majority of general practitioners are pleased to cooperate clinically in this management. All newly diagnosed patients with genital herpes are now being directly asked for their permission to involve their general practitioner in their care and we will continue to audit our experience of the uptake of this. A small proportion of patients continue on hospital prescriptions because of their concern about confidentiality.

BARBARA VONAU
SIMON E BARTON
BRIAN G GAZZARD
Department of HIV/Genitourinary Medicine, St Stephen's Centre, Chelsea and Westminster Hospital, 369 Fulham Road, London SW10 9NH

Correspondence to: Dr Barbara Vonau.

1 The Herpes Simplex Virus Advisory Panel. Sharing care in genital herpes—new guidelines for the GP/GUM interface. *Br J Sex Med* 1996;May/June:13–5.

Accepted for publication 17 February 1997

Hypospadias associated with the use of high dose megestrol acetate in an HIV infected woman

Megestrol acetate has been used to stimulate appetite and promote weight gain in patients with acquired immunodeficiency syndrome (AIDS) related cachexia and wasting.[1 2] We report a case of hypospadias associated with the use of high dose megestrol acetate during the first trimester of pregnancy.

Hypospadias is a congenital malformation, in which the urethral meatus forms proximal to its normal position, resulting from incomplete fusion of the urethral groove during fetal development.[3] (The normal process of fusion is brought about by androgens from the fetal testes during the first trimester of pregnancy.) Hypospadias is a relatively common abnormality, with a prevalence ranging from 1 in 300 to 1 in 1000 male births in the general population.[4 5]

Synthetic progestogens have been suggested as possible low risk teratogens for a range of congenital abnormalities.[6 7] While the association of hypospadias with the use of standard doses of synthetic progestogens during pregnancy has been described,[5 8] there have been no reports to date of birth defects associated with the use of high dose megestrol acetate.

Thirty women with human immunodeficiency virus (HIV) infection, and more than 10% weight loss, were enrolled in a study of weight gain using an oral suspension of megestrol acetate. Patients were randomised to receive either 400 mg or 800 mg of megestrol acetate per day for 24 weeks. A 28 year old HIV positive female participated in the study with the following chronology of events. At enrolment, she had had surgery 2 months earlier for an ectopic pregnancy with irregular menses, and her initial serum pregnancy test was negative. She was counselled regarding the necessity of using barrier method contraception. She started taking megestrol acetate but failed to attend for follow up clinic visits. Subsequently, pregnancy testing and ultrasonography demonstrated that she was 17 (SD 2) weeks pregnant. It was determined retrospectively that she had taken megestrol acetate, 400 mg per day, for 18 days from the 4th to the 7th week of pregnancy (by ultrasound dates). Her only other medication was zidovudine, 600 mg per day. At 38 weeks' gestation, she delivered by repeat caesarean section a live male infant, with normal Apgar scores, weighing 2633 g, with second degree hypospadias. The boy, now 7 months old and HIV negative, will require corrective surgery.

High doses of megestrol acetate in the first trimester of pregnancy may increase the risk of hypospadias. This warning appears in the drug manufacturer's prescribing information.[9] Caution needs to be exercised in prescribing megestrol acetate to HIV infected women with reproductive potential. *Repeated* counselling of patients on the use of adequate contraception and education of staff and patients regarding potential teratogenic effects of megestrol acetate should be stressed.

DAVID J FARRAR
IREENE AROMIN
SUSAN CU UVIN
TIMOTHY P FLANIGAN
MARIA D MILENO
Department of Medicine, The Miriam Hospital, Brown University School of Medicine, Providence, Rhode Island, USA

Correspondence to: Dr M D Mileno, Immunology Center, The Miriam Hospital, 164 Summit Avenue, Providence, RI 02906, USA.

1 Von Roenn JH, Armstrong D, Kotler DP, Cohn DL, Klimas NG, Tchekmedyian NS, *et al.* Megestrol acetate in patients with AIDS-related cachexia. *Ann Intern Med* 1994; **121**:393–9.
2 Oster MH, Enders SR, Samuels SJ, Cone LA, Hooton TM, Browder HP, *et al.* Megestrol acetate in patients with AIDS and cachexia. *Ann Intern Med* 1994;**121**:400–8.
3 Stock JA, Scherz HC, Kaplan GW. Distal hypospadias. *Urol Clin N Am* 1995;**22**:131–8.
4 Dawson C, Whitfield H. ABC of urology: common paediatric problems. *BMJ* 1996;**312**: 1291–4.
5 Aarskog D. Maternal progestins as a possible cause of hypospadias. *N Engl J Med* 1979; **300**:75–8.
6 Katz Z, Lancet M, Skornik J, Chemke J, Mogilner BM, Klinberg M. Teratogenicity of progestogens given during the first trimester of pregnancy. *Obstet Gynecol* 1985;**65**: 775–80.
7 Darling MR, Hawkins DF. Sex hormones in pregnancy. *Clin Obstet Gynecol* 1981;**8**: 405–19.
8 Allen TD, Griffin JE. Endocrine studies in patients with advanced hypospadias. *J Urol* 1984;**131**:310–4.
9 Megestrol acetate (Megace) product labelling and package insert.

Accepted for publication 3 March 1997

Lymphoedema of the genitalia secondary to skin tuberculosis: report of three cases

Lymphoedema of the genitalia due to lymphatic obstruction is generally caused by filariasis, at times by neoplastic changes, and rarely, by lymphogranuloma venereum or donovanosis.[1] We report its unusual occurrence in two patients with scrofuloderma and one with lupus vulgaris.

Case reports

CASE NO 1

A 25 year old woman with a 15 year history of recurrent swellings in the neck and groins had taken indigenous treatment with no relief. Later she had swelling of the vulva which brought her to the hospital. Examination revealed irregular scarring and few intermittently discharging sinuses over the submandibular and cervical areas. Multiple abscesses and sinuses were seen affecting the inguinal lymph nodes of both sides. The soft and swollen vulva showed vesicles, some of which had eroded. Systemic examination revealed no abnormality.

Investigations revealed a haemoglobin of 10 g/dl, white cell count 10·4 × 10⁹/l, differential—polymorphs 50, lymphocytes 34, eosinophils 26, erythrocyte sedimentation rate 40 mm in the first hour. The enzyme linked immunosorbent assay to detect HIV was negative. Mantoux test to 0·1 ml of purified tuberculin (Span Diagnostics, Surat, India) injected on the volar surface of forearm read 40 mm × 40 mm after 72 hours. *x* Rays of the chest and pelvis disclosed no abnormality. Biopsy from an active lesion was sent for histopathology and culture in Lowenstein-Jensen medium. The former revealed a thinned and ulcerated epidermis. In the dermis an acute inflammatory infiltrate was seen around a necrotic area. The deeper dermis showed few tuberculoid structures with epithelioid and occasional Langerhans giant cells surrounded by lymphocytes. No acid fast bacilli were demonstrable in Ziehl-Neelsen stained sections. *Mycobacterium tuberculosis* was isolated on culture. Oral antitubercular treatment (ATT) comprising rifampicin 450 mg/day, 300 mg/day and pyrazinamide 750 mg twice daily was started. Significant improvement was seen after 2 months; pyrazinamide was stopped and the first two were continued. After 3 months the vulval swelling had decreased. She was advised to continue regular treatment but did not report to the hospital again.

CASE NO 2

A 30 year old beggar had occasionally discharging inguinal lesions of 10 years' duration. They had started on the right side and spread over a period of time. There was no history of pulmonary tuberculosis. He had later noticed an increase in scrotal size. Examination revealed fluctuant areas and partially healed sinuses involving the inguinal lymph nodes of both sides. Healed areas were connected by thick scars extending into the suprapubic area. There was scanty to moderate discharge from the sinuses. The scrotum and penile skin were stretched and oedmatous (fig). The perianal region appeared normal.

Routine blood and urinalysis, and *x* rays of the chest and pelvic area were within nor-